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## General and Applied

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**Sleep Deprivation: Changes in Performance and Physiological Indicants of Activation**

By

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Psychological Monographs: General and Applied

# SLEEP DEPRIVATION: CHANGES IN PERFORMANCE AND PHYSIOLOGICAL INDICANTS OF ACTIVATION[1]

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EARLY investigations of sleep deprivation arose mainly from an interest in the influence of fatigue on performance. Except for a few studies such as the remarkable account of a 10-day vigil of a man who believed sleep to be unnecessary (Katz & Landis, 1935) this early work was rather inconclusive with respect to the effects of sleep loss on performance. A review of the early literature may be obtained from Edwards (1941), Katz and Landis (1935), Kleitman (1939), and Laslett (1928).

Not until recently (Kornetsky, Mirsky, Kessler, & Dorff, 1959; Wilkinson, 1958, 1959; Williams, Lubin, & Goodnow, 1959) have really adequate methodology and statistical treatment of results been applied to the problem of sleep loss and performance in human subjects (*S*s). Wilkinson's excellent studies are particularly noteworthy for the large number of *S*s employed and for the variety of the situations studied. One of his most significant findings appears to be the hitherto unrecognized importance of feeding back information to the sleep-deprived *S* about the scores that he is making. Without such feedback in a serial reaction test, performance was considerably impaired under the condition of sleep deprivation, whereas with feedback this impairment was practically eliminated. This finding is well established in two separate studies by Wilkinson (1958, 1959), and in the latter study, Wilkinson (1959) concludes that although inadequate incentive tends to yield poor performance even in normally rested persons, the need for adequate (and frequent) incentive becomes disproportionately great in those who have been sleep deprived.

*Animal Studies.* Employing rats as *S*s, the effects of enforced wakefulness on learning and performance in a water maze were investigated by Bunch, Frerichs, and Licklider (1938), and by Licklider and Bunch (1946). The 1938 experiments with periods of enforced wakefulness of 8, 16, 24, and 48 hours, produced rather surprising results, showing that in the case of all learning criteria (trials, time, and errors) the wakeful rats were superior to the controls. In the 1946 study, the animals' sleep was restricted to 4 hours per day, one group of wakeful rats undergoing 123 days of experimentation and a second group of wakeful rats undergoing 73 days of experimentation. Sleep deprivation again appeared to have facilitative effects although performance rather than learning appeared to be affected, and the authors stated (p. 346): "the better performance of the wakeful rats is largely to be accounted for in terms of their more aggressive behavior." In their conclusions the authors stressed

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that the restriction of the animals' sleep made them "highly irritable and highly reactive."

Nauta (1946), who produced sleeplessness in rats by making lesions in the preoptic nucleus, reported that after a period averaging 3 days "the exhausted animals fell into a state of coma which soon ended in death" (p. 303). It appears that sleeplessness produced by lesions may accelerate collapse, because Licklider and Bunch (1946) reported that rats in good health collapsed in 4 to 18 days without sleep. Before Nauta's rats collapsed, however, they too showed irritability, restlessness, and vigorous reactions to minor stimuli. Nauta likened some of this behavior to sham rage.

*Sensory Thresholds.* Several studies have been concerned with determinations of sensory thresholds under conditions of sleep deprivation. The length of the deprivation period varied from study to study, and in certain studies thresholds were determined after different periods without sleep. Cooperman, Mullin, and Kleitman (1934) in a study of cutaneous sensibility carried out with von Frey hairs, found that reports of pain occurred with weaker stimulation during a vigil of 60 hours. From their article it appears that this increased sensitivity to pain was not observed during the determinations made after 24 hours of sleeplessness; and this finding is in agreement with that of Schumacher, Goodell, Hardy, and Wolff (1940) that 24 hours' sleep deprivation produced no change in the thermal pain threshold. Except for the increased sensitivity to pain, and decreased sensitivity to sour taste (Furchtgott & Willingham, 1956), changes in sensory thresholds have not been observed following sleep loss. Negative findings were obtained with touch by Cooperman et al. (1934) after approximately 60 hours' deprivation, with audiometric tests by Goodhill and Tyler (1947) after 50 and 100 hours' deprivation, and with limens for salt and sweet taste by Furchtgott and Willingham (1956) after 24-, 48-, and 72-hour periods of sleep deprivation.

*Electroencephalographic and Other Physiological Changes.* EEG changes in the sleep-deprived *S* were, as far as we know, first studied by the group working with Kleitman. Among other things, they noted that when the *S* was engaged in a mental task requiring his concentration there was "a great discharge of beta waves which replaced the delta rhythm" (Blake, Gerard, & Kleitman, 1939, p. 55).

Tyler, Goodman, and Rothman (1947) investigated the effect of sleep deprivation on the percentage of time that each frequency occupied in the EEG tracing. These workers were careful to exclude from their measurements any segments of records taken when the *S* was suspected of dozing. The authors concluded that at 75 hours of sleep deprivation there was a shift in the percentage of time occupied by the various frequencies such that less time was occupied by frequencies around 9–10 cps, and more time was occupied by the higher frequencies (11–17 cps and 18 cps and higher). Control recordings were taken 3 days prior to the experimental period, and were compared with recordings taken under three separate sleep deprivation conditions: 50, 75, and 100 hours, with a different group of four *S*s serving in each of the three conditions. The authors concluded that the decrease in 9–10 cps activity and the corresponding increase in the higher-frequency activity was associated with the effort exerted by the *S*s in staying awake. This conclusion was based on their observation that similar changes in the EEG were found to occur while the *S*s were engaged in problem solving.

Bjerner's (1949) EEG study was mainly concerned with a different aspect of the sleep-deprivation phenomena. Instead of excluding EEG measurement during brief periods of sleep as Tyler et al. had done, Bjerner studied these inadvertent "catnaps." His five *S*s performed serial reactions with an apparatus specially designed for this study. The major part of the data analysis consisted in locating instances of gross lapse in performance (delayed actions) on the graphic tracings of performance, and then marking off the corresponding parts of the EEG and heart rate tracings for measurement. In this EEG analysis Bjerner concerned himself only with the number of

alpha waves counted (*a*) during the period of delayed action, (*b*) for the 3 seconds before, and (*c*) the 3 seconds after the delayed action. Using the 3-second periods before and after delayed actions as basis for comparison, Bjerner found a significant drop in the number of alpha waves during delayed actions.

It is important to recognize that Bjerner's procedure was radically different from that of Tyler and his co-workers whose measurements were confined to parts of the vigil when the experimenters (*E*s) were certain that the *S* was not dozing. Bjerner's observations, on the other hand, were confined to periods when the *S* had dozed off during the performance of the serial reaction test. Bjerner's protocols actually noted the occurrence of snoring during some of the periods of delayed actions when the tracings to be measured were being taken. Accompanying the significant fall in number of alpha waves from the EEG during delayed actions was a significant transient fall in heart rate.

In a recent investigation Armington and Mitnick (1959) reported a progressive reduction in the amount of alpha (8–12 cps) activity with sleep deprivation. In the interpretation of these results, Armington and Mitnick referred to the work of Stennett (1957a) in pointing out that low alpha may be indicative of either a high or low level of activation, and they interpreted the reduction of alpha in their experiment as being due to drowsiness or a low level of activation.

Finally, an observation concerning the skeletal motor system may be mentioned. In a study in which he and his wife served as alternative *S* and *E*, Freeman (1932) noted increased quadriceps tonus on days following serious sleep loss.

*Hallucinations.* Numerous authors have mentioned hallucinatory experiences in sleep-deprived *S*s, but unfortunately the number of well described cases in which hallucinations definitely occurred is very small. With regard to hallucinations Tyler's (1947) investigation is unusually detailed. Of the total number of 588 *S*s studied, 444 *S*s were deprived over 60 hours (with 275 *S*s deprived as much as 112 hours). Some of the results of this study were complicated by administration of drugs (benzedrine sulphate and barbiturates) and by an extremely strenuous program of exercise. After stating that in almost all cases the psychological changes observed were only mild ones, Tyler wrote as follows:

> In a few cases the psychological changes which occurred were not so mild. These developed during the first 48 hours of sleeplessness and while they were still receiving placebos. The first such instance occurred just before dawn of the third day during a 112-hour experiment involving 35 men. This subject became extremely aggressive, developed delusions of grandeur (imagined himself on secret missions for the President, etc.), started unprovoked fights, became unmanageable, and had to be forcibly restrained. He was put to bed and slept through the entire day. On awakening he had no recollection of the events of the previous night. An examination of his record disclosed no indication of any psychotic tendencies during his training period; he was well liked, and considered quiet and reserved by his friends.
>
> Six other such cases occurred during the course of these studies: two were characterized by extreme irritability and aggressiveness requiring restraint; four cases by persistent auditory and visual hallucinations of such a nature that it was deemed desirable to have the men drop out. As in the case described above, none of these six men had a previous history of psychotic tendencies and, following sleep, all symptoms and all recollection of the preceding events disappeared (pp. 258-259).

As far as we know there has been no systematic study of physiological functions other than EEG and heart rate in conjunction with performance under conditions of sleep deprivation. Furthermore, with the possible exception of the work of Tyler et al. (1947), there have been no investigations made of physiological changes through the vigil under conditions which *force* the *S* to suppress sleep and to keep performance at or near the level of which he is capable in the absence of sleep loss.

The experiments to be described presently were designed to fill this gap. They contained feedback-of-information features to counteract lapses in the *S*'s tracking performance due to brief periods of sleep. Wilkinson's (1958, 1959) experiments showing the importance of feeding back information to a sleep-deprived *S* about his scores were noted earlier. Although the results of Wilkinson's experiments were not

known during the design of this study, the *E* communicated with the *S* at the end of every trial and informed him what his score was on that trial and how that score compared with his best score up to that time. Two other means of informing the *S* concerning his errors (heaters on his legs that approached painful intensity with accumulation of gross errors, and the intensity of the tone in the earphones which was proportional to the *S*'s error) were employed to further decrease the liability of the *S* to lapses caused by a drift toward sleep.

To summarize, the main purpose of the present investigation was to observe changes in physiological and performance measures as a function of sleep deprivation under conditions designed to be highly alerting, viz., with the *S*s instructed to guard against falling asleep, even for brief intervals, and with various techniques involving continuous feedback of information concerning performance employed to keep alertness at a high level.

Data relevant to the matter of changes in pain thresholds with sleep deprivation were collected in the course of these experiments and finally, the relation of body sway to sleep deprivation was also studied.

## METHOD

### *Subjects*

Three young healthy male *S*s were employed in the experiments. $S_1$ was a 28-year-old clinical psychologist on our laboratory staff, $S_2$ a 26-year-old graduate student assigned to our laboratory for the summer, and $S_3$ a 25-year-old third-year medical student who had volunteered to serve as *S* after hearing about our experiments. Prior to his learning sessions on tracking before the vigil, this *S* had not been in our laboratory. $S_1$ and $S_2$ had served in pilot studies with very long sessions and they were highly practiced in tracking. All three *S*s were well paid for serving, and they all assumed their responsibilities with quite evident seriousness.

Medical examination prior to the experiment established that all were in excellent physical condition and, during the experiments, the *S*s were examined regularly by a physician. Except for a mild digestive upset in $S_1$ on the last day of his vigil (which, in the opinion of the physician did not require stopping the experiment) the experiments were uneventful from the medical point of view. $S_1$ was given one tablet of gelusil and one tablet (50 mg.) of gravol (dimenhydrinate) following breakfast after which his symptoms of mild nausea disappeared within an hour. At lunch time the same symptoms which were reported in the morning (mild nausea and lack of interest in food) returned. Approximately one-half of his lunch was left. These symptoms disappeared within one-half hour following administration of the same medication.

Physical examination revealed a hearing loss in $S_3$ which was subsequently investigated more closely because of its importance for auditory tracking. From comparisons of the *S*'s lower limen at 1000 cps with those of three observers with unimpaired hearing, it appeared that the *S*'s hearing loss for this frequency was in the neighborhood of 5 db. To compensate for this loss, the level of the tracking tone was raised approximately 5 db.

### *Apparatus*

#### *Tracking Apparatus*

With certain modifications, the apparatus used in this study was identical with that previously described (Davis, Stennett, & Quilter, 1957). For this study, however, the directional cue was eliminated; hence an error in either direction produced a 1000-cycle tone in the earphones worn by the *S*. Seated comfortably in a semireclining position, the *S* was required to grasp and turn a knob with his right hand in such a manner as to prevent a 1000-cycle tone from appearing in the earphones. For purposes of EEG recording the *S* was blindfolded. The tracking task was a simple one in that it was only necessary for the *S* to turn the knob slowly to the left until he reached a mechanical stop and then let the knob return slowly to the right against the other stop. This pattern was repeated cyclically. As a further means of being sure that the *S* was constantly alert, he was asked to press his right foot down on a pedal each time that he came to one of the mechanical stops. By pressing down on the foot pedal the *S*

closed a microswitch which caused a pip to appear on the polygraph tracing.

Time-off-target (TOT) was recorded by direct-reading SET clocks and also graphically by means of an integrator with a 4-sec. discharge rate. The instrument was calibrated such that 1 sec. off-target produced a deflection equal to 1 cm.; TOT was registered whenever the amount of error was approximately equal to or greater than 0.58°. Using the same kind of integrator, distance-off-target (DOT) in fractions of a degree was also recorded graphically. The amplitude of deflection per degree off-target was controlled by attenuators in the error-recording circuits. The attenuator settings were varied in accordance with the *S*'s level of performance. From calibrations, special scales were constructed for measurement of the DOT tracings.

### *Physiological Recording Apparatus*

For recording physiological tracings and error data, three chart drives with ink-writing galvanometers were used. Chart drive no. 1 was part of a custom-built Edin EEG-EMG which recorded the primary tracings of EEGs and electromyograms (EMGs) (paper speed: 25 mm. per sec.). Electronic integrators, employing the condensor charge-discharge principle and connected in parallel with the galvanometers of this EEG-EMG unit, summated the muscle potentials over successive 4-sec. periods. The spike tracings from these integrators were recorded on chart drive no. 2 (paper speed: 1 mm. per sec.). High-pass filters designed to exclude frequencies below 20 cps were used for EMG integrations (where the modal frequency is of the order of 40 to 50 cps). For quantification of the EEG data two band-pass filters with fairly sharp cut-offs at the upper and lower limits of the 2–4 cps and 8–12 cps frequency bands were used in connection with integrators.

Chart drive no. 3 was used to record heart rate, respiration, systolic blood pressure, palmar conductance, DOT, and TOT. Heart rate was recorded by means of a modified Edin electrocardiotachograph (ECTG), and respiration was recorded with a Phipps and Bird pneumograph and recording tambour. An attempt was made to record systolic blood pressure continuously by means of an instrument copied with certain modifications from the design of Burns, Elliott, and James (1955). Unfortunately, however, the tracings from $S_3$ were not satisfactory and after Session 6, they were discontinued in the case of this *S*. Method of recording and measuring palmar conductance was similar to that described by Stennett (1957b).[3]

[3] Some important details of the apparatus for recording palmar conductance and some advances in recording technique are described in an unpublished paper by Malmo and Davis.

Bipolar sponge electrodes, soaked in electrode jelly and attached to the *S* by lastonet bands, were used for all EMG and EEG placements. Lead placements for $S_1$ and $S_2$ were as follows (EMGs from neck and biceps muscles were omitted for $S_3$): For the forehead-muscle recording, electrodes were placed 2 in. from the midline (4 in. from each other) and approximately 1 in. above the eyebrow. In recording from the muscles on the left side of the neck, the second cervical spine (found by counting from the prominent seventh cervical spine) was located and marked. A point 1¾ in. outward from the second cervical spine (perpendicular to the backbone) was marked for the placement of the first electrode. The second electrode was placed 1¾ in. downward from the first electrode and ¾ in. to the left of the midline.

For recording from the pronator teres muscle, a line was first constructed across the ventral surface of the arm from medial epicondyle to lateral epicondyle. The center of the first electrode was placed 1 in. medial to the midpoint between the two bony landmarks. The second electrode, its center 2 in. from the first, was the vertex of an equilateral triangle whose base lay along the elbow crease and whose vertex in the longitudinal plane lay along the midline of the arm (approximately 1¾ in. directly below the cubital fossa). In this study, recordings were taken from the left ("inactive") arm as well as from the right arm that was engaged in tracking.

In placing the first right-biceps lead, a point was marked one half of the distance from the anterior fold of the axilla to the cubital fossa, and the center of the first electrode was placed 1 in. proximally from this point (on the line between the two landmarks). The center of the second electrode was placed on the same line 2 in. distally from the center of the first electrode.

In placing the first electrode for the left leg, the distance from the head of the fibula to the lateral malleolus was measured, and the point which bisected this line was marked. The center of the first electrode was placed at this point. The center of the second electrode was placed on the same line, 2 in. proximal to the first.

Placements of all muscle leads except pronator teres are illustrated in a mimeographed manual by Davis (1952), and other technical points are dealt with in this manual. For EEG recording, bipolar sponge electrodes soaked in electrode jelly were attached to the *S* by lastonet tie bands, and these electrodes were placed two thirds of the distance from nasion to inion, and 3 cm. from the midline on each side. ECTGs were recorded by means of electrodes placed on contralateral limbs.

### *Heat-Pain Stimulation as a Means of Increasing the Subject's Motivation*

In order to increase the *S*'s incentive to track well throughout the vigil, heater coils were connected to the legs which in turn were wired into

a circuit activated by the error relay. Whenever the error score became disproportionately high, enough heat was generated by these coils to become actually painful. A detailed description of this apparatus has previously been published (Surwillo, 1958).

### *Other Apparatus*

During part of the time that the *S* was not tracking, pain-threshold determinations were made and measurements of postural sway were recorded. The apparatuses employed in these two procedures were as follows:

*Modified Hardy-Wolff Pain-Threshold Apparatus.* Pain-threshold measurements were made with laboratory-constructed apparatus essentially the same as that used by Andrews (1943). The standard 3-sec. period of stimulation was provided by a shutter controlled in turn by a Hunter timer which was specially calibrated in our laboratory.

A 500-w., 120-v. Westinghouse T20 clear projection lamp (C13 construction) with medium, prefocused base, supplied the stimulus. This lamp was operated in the base-down position from the AC line through a voltage stabilizer and controlled by a Variac transformer. Each stimulus was measured by a wattmeter connected in the lamp circuit.

The lamp was mounted inside a bullet-shaped metal housing with an aperture of $\frac{1}{16}$-in. diameter in the nose. Two Bausch and Lomb lenses, 165.1 mm. in diameter, 31.9 mm. in thickness, and with 254.0-mm. (single lens) focus were combined (flat surfaces outward), and placed between the lamp and the aperture so that the focal length of the combined lenses was approximately 6 in. Two rounded plastic stops, 1 in. long and $\frac{3}{4}$ in. in diameter at their ends, protruded from the nose.

A very large area of the *S*'s forehead was blackened with Higgins drawing ink and allowed to dry well. After he had been informed concerning the threshold-taking procedure, the *S* placed his forehead against the stops in such a way that the focused light would fall on the blackened portion of his forehead. The *E* turned the wattage down to a low value and closed a switch which activated the Hunter timer. The timer caused the shutter to open and close allowing the light to fall on the *S*'s forehead for exactly 3 sec. For the first stimulation the wattage selected was one which almost always elicited the response "warm" from the *S*. Following his verbal response, the *S* withdrew his head from the stops while the *E* increased the Variac setting to a point where the wattmeter read 20 w. higher than on the previous stimulation. After 15 to 20 sec. the *S* was asked to place his forehead against the stops again, shifting the position slightly from where it had been on the previous stimulation. When the *S* was in position and had stated that he was ready, the *E* presented the second stimulus for 3 sec. and recorded the *S*'s response. This procedure was repeated with the *S* returning his forehead to the stops each time so that the area of skin stimulated was not the same on two successive occasions, and with the *E* advancing the intensity by 20 w. from stimulus to stimulus until the verbal response of pain was elicited from the *S*. It had previously been explained to the *S* that the procedure was by no means a test of his ability to endure pain, but rather was a means of determining what intensity of stimulation was required for him to perceive the first trace of pain. The *E* also explained that it was especially important for the *S* to concentrate during the latter part of stimulation because the slight pricking or stinging feeling, signifying that the pain threshold had been reached, appeared at the very end of the 3-sec. stimulation period. At least two threshold determinations were taken at each sitting but three determinations were invariably made unless the first two threshold values were extremely close.

All *S*s were given practice on the threshold-taking procedure and had thresholds taken prior to the vigil. During the vigil, thresholds were taken during the midafternoon of the first, second, and third days, and early in the morning (between 5:00 A.M. and 6:30 A.M.) of the second and third days. Pain thresholds were taken following sleep after the vigil in all cases. In consideration of the *S*'s convenience, however, it was possible to test only $S_2$ on the same morning after his sleep following the vigil.

*Ataxiameter.* This apparatus was an adaptation of Hull's (1933) Postural Movement Recorder. A clip with thread attached was fastened to the back of the *S*'s collar. This thread was led through a protective metal tubing on to a pulley and thence attached to an upright metal pointer whose base was held in a metal track. Next, the thread was led over a second pulley whence it ran vertically. A weight was attached to this end to keep the thread taut. When the *S* leaned forward, the pointer moved in its track to his right; when he leaned backward, the pointer moved to his left. The *S* was positioned initially so that the pointer rested midway along the track, pointing to zero on the scale. This scale, which extended 30 cm. to the right and 30 cm. to the left of the zero point, was marked off into major divisions of 1 cm. which in turn were quartered. In addition to the red pointer attached to the thread, two blue pointers were fitted into the track, one on each side of the red one. The blue pointers were used to record the maximum excursions of the red pointer (maximum forward and backward movement of the *S*).

The *S* stood with eyes closed and with feet in "V" position, at an angle of 30° with each other. After being instructed to keep his body as still as possible the *S* stood for 2 min., rested for 30 sec., and then stood for 2 min. again. The scale location of the red pointer was recorded every 10 sec. together with the maximum left and right (blue pointer) readings at the end of each 2-min. period.

TABLE 1

SCHEDULE OF TRACKING SESSIONS DURING VIGILS

| Session No. | Time Scheduled | Day |
|---|---|---|
| 1 | 11 A.M. | Tuesday |
| 2 | 3 P.M. | |
| 3 | 9 P.M. | |
| 4 | 3 A.M. | Wednesday |
| 5 | 11 A.M. | |
| 6 | 3 P.M. | |
| 7 | 9 P.M. | |
| 8 | 3 A.M. | Thursday |
| 9 | 11 A.M. | |
| 10 | 3 P.M. | |

Ataxiametric sessions were scheduled two hours apart throughout the vigils, and except for some occasions when delays occurred, these sessions were kept fairly close to schedule.

### *Procedures*

From the *S*'s point of view the vigils were structured mainly by the 10 1-hour tracking sessions (see Table 1) and by meals. Five-min. tracking trials were employed, with 1-min. rest intervals between trials. There were 10 such trials in each experimental session. After each trial the *E* informed the *S* what his score was on that trial and how that score compared with his best score up to that time. In the case of $S_2$ and $S_8$, at the end of every session, full recordings were made during a 5-min. rest period in which the *S* remained seated in the chair under careful observation to make sure that he remained awake all through the period. In drawing up the master plan for all activities during the vigil, two hours prior to the times shown in Table 1 were allowed for precalibrations, the attachment of electrodes and other pickups to the *S*, and presession trial of all recordings. One hour was the minimum time for a session, but it was expected that most sessions would run slightly longer due to occasional stops to adjust or change faulty electrodes, or to make minor adjustments in the apparatus to ensure first-class recordings. A whole hour after the experimental session was set aside for removal of electrodes and attachments from the *S*, and for postcalibrations. Certain electrodes that could be worn for long periods without discomfort to the *S* were left on. Despite the large number of measures recorded it was possible to keep rather close to this schedule.

Meals from the institute's Diet Kitchen[4] were served to the *S* in the laboratory room. Breakfast was at 8:00 A.M., lunch at 12:30 P.M. and supper at 5:30 P.M. In addition to regular meals, morning coffee was served at 10:00 A.M. and light snacks were served at 2:00 P.M. and 10:00 P.M. The *S* remained in the same laboratory room throughout the vigil except for trips to the washroom and to a room adjacent to the laboratory to make an occasional telephone call. The experimental room was windowless and in order to prevent the *S* from perceiving the changes from daylight to darkness outside, he was blindfolded and led by one of the laboratory assistants whenever he went to the other two rooms. No attempt was made to conceal the time of day from the *S*; the purpose of the blindfold was merely to avoid giving the *S* opportunity to perceive changes in illumination from the sky. It has occasionally been reported that the subjective feeling of fatigue during a vigil is lessened at daybreak when the *S* perceives the sky becoming light.

Regularly following each tracking session two graphic rating scales were administered. In each instance the *S* was requested to place a vertical line somewhere along an unbroken horizontal line that was 20 cm. in length. The question guiding the rating of the first scale was, "How much effort did you have to put in the task in order to do as well as you could?" The guide phrases "considerable amount of extra effort," and "normal amount of effort" appeared at opposite ends of the horizontal line.

In the case of the second scale the question was, "How do you feel now?" Five guide phrases evenly spaced along the horizontal line were as follows: "very tired," "tired," "as usual," "fresh," and "very fresh." The order of the guide phrases from left to right was reversed on alternate presentations of each scale.

Soon after supper each evening and between midnight and 1:00 A.M. on Wednesday and Thursday, the *S* took a variety of tests administered by clinical psychologists. The purpose of these tests was mainly to keep the *S* actively engaged in order to prevent sleep. Between these planned activities and procedures the *S* was free to read, play games, or do whatever he pleased ($S_1$ spent much of his free time playing his guitar). The *S* was under constant observation by one member of the laboratory or another, and care was taken to have at least one well-rested laboratory assistant on duty at all times. At no time was the *S* left alone, even for a few minutes.

### *Treatment of Data*

#### *EEG*

The EEG tracings recorded on one channel of the Edin instrument were used for monitoring purposes. Band-pass filters and integrators were employed to quantify the EEG tracings in the two frequency ranges of approximately 2–4 cps and 8–12 cps. The integrated data were separately

[4] It is a pleasure to record our gratitude to the dieticians and to other personnel of the Diet Kitchen of the Allan Memorial Institute for their important contribution to this study.

recorded as deflections every 4 sec. (0.1 cm/sec paper speed).

Using all of the integrator deflections, the mean voltage per min. was determined by accumulating the number of millimeters in each successive series of 15 deflections dividing by the number of deflections (usually 15) and by reference to the specially calibrated ruler for the gain used in recording, reading off the corresponding microvolt value.

For each frequency range (2–4 cps and 8–12 cps) five mean 1-min. microvolt values were obtained from each tracking trial and 50 such values were obtained from one entire tracking session, providing no deflections were monitored out. The original tracing was carefully examined in order to ensure that no spike was measured if it covered a part of the tracing in which some artifact occurred. Where available (in the case of $S_2$ and $S_3$) the data for the 5-min. rest periods at the end of the sessions were treated in the same way as the data for the tracking trials.

Following measurement of the EEG integrations, the separate measurements were combined in various ways. The 250 1-min. mean microvolt values from the tracking trials in the first five tracking sessions were combined to yield a mean for the first half of the vigil, and similarly the mean microvolt value for tracking sessions in the second half of the vigil was also obtained. Mean microvolt values were also computed separately for each tracking session, for each 5-min. tracking trial, and for each 5-min. rest period.

### *EMG*

With the exception of the tracings from the right arm, every sixth deflection was measured, commencing with the sixth deflection, such that 12 deflections in all were measured for every trial. For muscles of the right arm, in order to keep the task-induced tension approximately the same from measurement to measurement, the following deflections were measured: Numbers 4, 12, 19, 27, 34, 42, 49, 57, 64, and 72.

As in the case of EEG and other physiological measures, the main comparison was between mean values from the first half of the vigil with mean values from the second half.

### *Palmar Conductance*

Measurements of palmar resistance were taken regularly every 15 sec. throughout each trial, commencing at the very beginning of the trial, so that there were 21 measurements per trial. Resistance values were immediately converted to conductance in micromhos with a table of reciprocals. All calculations and statistics were performed with conductance values as recommended by Woodworth and Schlosberg (1954, p. 140).

### *Respiration*

Number of respirations counted to the nearest quarter respiration was obtained for the first 2½ min. of each trial, for the last 2½ min., and for the whole trial.

### *Heart Rate*

Heart rates were determined from the ECTG tracing at 15-sec. intervals, these measures coinciding temporally with the measurements of palmar conductance.

### *Tracking Error Scores*

*TOT.* The clock readings showed total TOT for each whole trial. Since the 4-sec. graphic integrations of TOT were available for fractionations involving parts of trials each deflection was measured with a rule calibrated directly in sec. Both TOT values were treated to yield a mean TOT score per trial for the first half of the vigil versus a mean TOT score per trial for the second half of the vigil. TOT scores were also obtained for the first half and last half of each trial.

*DOT.* Each 4-sec. integrator deflection was measured with a rule calibrated in degrees. Mean DOT scores for the first half and second half of the vigil were determined. DOT scores were also obtained for the first half and last half of each trial.

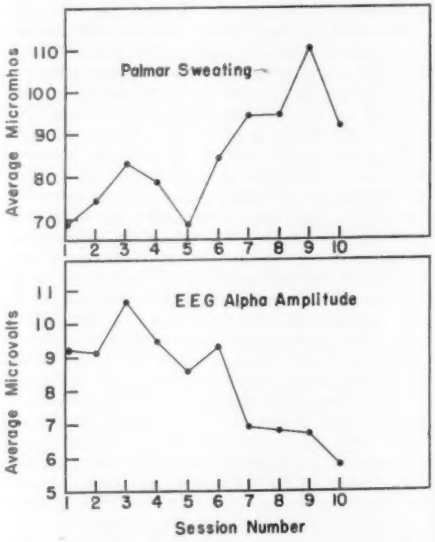


FIG. 1. Mean levels of 8–12 cps EEG and palmar conductance throughout vigils lasting approximately 60 hours. (Each point on curves represents mean value for 1-hour tracking session, combining the values for all three *S*s. Approximate time corresponding to each session number is given in Table 1.)

## RESULTS

### *EEG, Palmar Conductance, and Respiration*

*Progressive EEG Changes during the Vigil in Relation to Concomitant Changes in Palmar Conductance and Respiration.* The lower graph in Fig. 1 presents data for EEG alpha quantified by means of the 8–12 cps band-pass filter. The upper graph in Fig. 1 presents data from measurements of palmar conductance. Each point on the curves represents the mean value for one entire tracking session. Figure 2 presents the corresponding data from respiration rate. Table 2 presents a comparison between mean values for the first half of the vigil versus mean values for the second half in the case of each $S$, and the $p$ values for the differences obtained.

Table 2 indicates that all three of these physiological measures showed reliable change for each $S$ during the vigil, with palmar conductance and respiration higher during the last half of the vigil and amplitude of 8–12 cps EEG lower during the last half as compared with the first half of the vigil.

EEG data quantified by means of the 2–4 cps band-pass filter also yielded significant changes for all three $S$s from first to last halves of vigil. $S_1$ and $S_3$ showed drops in amplitude from 5.3 to 3.9 ($p < .001$) and from 3.3 to 3.0 ($p < .01$) mean microvolts, respectively, while $S_2$ showed a rise from 3.7 to 4.0 mean microvolts ($p < .001$).

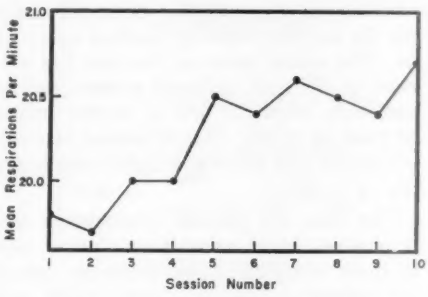


FIG. 2. Mean rates of respiration throughout the vigils. (Points on curves were obtained similarly to those in Fig. 1.)

*Comparison of Within-Trial Changes: A Search for Lowered 8–12 cps EEG ("Alpha") Associated with Possible Drowsiness and Shift toward Sleep.* Accompanied by a concomitant rise in palmar conductance and respiration, the finding of a progressive fall in 8–12 cps EEG activity is strong evidence that our $S$s were very much awake and alert throughout the tracking sessions (Stennett, 1957a). However, to investigate this matter more closely, the data were subjected to further analysis.

An inspection of the 8–12 cps EEG data revealed that amplitude tended to fall during the trial, and this tendency seemed more marked in the second half of the vigil than in the first. We present the following analysis by $S$s.

$S_1$: The mean difference between first and second halves of trials for the first five tracking sessions in the vigil was $-0.16$ $\mu$v.,

TABLE 2

CHANGES IN EEG 8-12 CPS AMPLITUDE, PALMAR CONDUCTANCE, AND RESPIRATION RATE DURING VIGIL

| | $S_1$ | | | | $S_2$ | | | | $S_3$ | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1st Half of Vigil | 2nd Half of Vigil | Diff. | $p$ | 1st Half of Vigil | 2nd Half of Vigil | Diff. | $p$ | 1st Half of Vigil | 2nd Half of Vigil | Diff. | $p$ |
| 8-12 EEG ($\mu$v.) | 12.4 | 9.3 | −3.1 | <.001 | 6.8 | 5.1 | −1.7 | <.001 | 9.1 | 7.0 | −2.1 | <.001 |
| PC ($\mu$ mhos.) | 49.6 | 64.7 | 15.1 | <.001 | 51.9 | 81.2 | 29.3 | <.001 | 113.2 | 139.1 | 25.9 | <.001 |
| Resp. (per min.) | 19.16 | 19.57 | 0.41 | <.05 | 21.34 | 22.18 | 0.84 | <.001 | 19.46 | 19.83 | 0.37 | <.02 |

whereas the corresponding mean difference for the last five tracking sessions was 1.64 $\mu$v. The minus value for the first five sessions of the vigil indicated a mean rise in amplitude from the first to second halves of tracking trials. The difference between −0.16 and 1.64 $\mu$v. was a highly significant one ($p < .001$).

The data for palmar conductance and respiration were treated in a similar way as those for EEG. The difference values for palmar conductance were −1.08 and −0.28 for the first five sessions and the last five sessions, respectively. As is well known, palmar conductance characteristically falls during a trial of almost any kind of activity. The data indicated, however, that the fall was significantly less ($p < .05$) in the latter part of the vigil than it was in the first part of the vigil. Corresponding difference values for respiration rate were 0.51 and −0.29 for the first five and last five sessions, respectively. This difference was reliable at the .01 level, and indicated that in the last five sessions there was a shift in the direction of slowed respiration in the latter halves of the tracking trials. Inasmuch as this finding was the reverse of that from palmar conductance, the respiration tracings were studied. Careful examination revealed an irregularity in this *S*'s breathing pattern which seemed to be more prominent in the latter part of the vigil than in the first part. This irregularity consisted in the occurrence of deep breaths which had the effect of reducing the respiration rate. In the latter part of the vigil this *S* took more deep breaths than in the early part of the vigil. Thus in the first three sessions there were only 4 large respirations (two times the height of adjacent respirations and at least 2 cm. wide at the base) compared with 42 such deviating respirations observed in the last three sessions. The breakdown for first and second halves of these trials were 0 and 4, 11 and 31, for the two fractionations (Sessions 1–3 and 8–10), respectively. It would appear, therefore, that the larger number of these deviating respirations in the latter half of trials late in the vigil may have been responsible for the apparent reversal between the data from respiration and those from palmar conductance.

$S_3$: This *S* also showed greater within-trial fall of 8–12 cps EEG amplitude later in the vigil. The difference values were 0.82 and 1.76 and the difference between these two values was significant at the .05 level. However, neither palmar conductance nor respiration yielded significant differences when treated in a similar way.

$S_2$: Data from this *S* failed to show a significant EEG difference when fractionated in the way described. However, error scores (TOT and DOT) showed significant differences when treated in this way. For TOT the difference values were −3.61 and +1.87 for

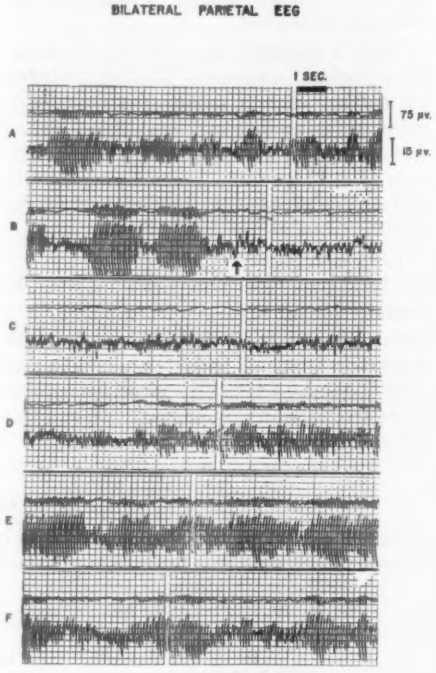


FIG. 3. Apparent disappearance of EEG alpha from the primary EEG tracing during brief episode of sleep in a young male *S* during tracking in an unpublished study by Malmo. (Search for similar phenomenon in the records of the present investigation failed. The arrow in this figure and those in Fig. 4 indicate the same moment in time.)

first and last parts of vigil, respectively (the plus score of 1.87 indicating a predominance of increasing error in the second half of trials), and the difference of 5.48 between these two values was highly significant (.001 level). For DOT the corresponding values were −0.009 and 0.003 and the difference of 0.012 between these two values was significant at the .05 level. Error scores from the other two *S*s were treated in this fashion but failed to yield reliable differences in either case.

Finally, it should be mentioned that a similar analysis was carried out on the heart rate data from the three *S*s but failed to yield any significant differences.

*Tracings Illustrating Transition from Waking to Sleeping State.* Finally, a careful search of the tracings was made for the purpose of locating instances of EEG changes characteristic of the kind which occur during a brief episode of sleep. This search did not reveal a single such instance.

In order to illustrate precisely what we were searching for, some tracings from an unpublished experiment by Malmo are reproduced in Fig. 3 and 4. These tracings were cut from Trial 9 of a series of tracking trials designed to bring the *S* to a practice level. The EEG lead placements were the same as those in the present study and the tracking apparatus was similar to the one described in the Methods section. As these tracings were being recorded, the *E* suspected from the pattern of changes occurring that the *S* had fallen asleep briefly during Trial 9. After the trial had been completed the *S* admitted upon questioning that he had indeed "dozed off."

Inspection of the record in Fig. 3 reveals that for a period of approximately 20 sec. there was virtual disappearance of the high-amplitude rhythmical waves with frequency close to 10 cps. This change is also seen in the integrator deflections in the upper part of Fig. 4 (second line). The deflections for the 17–30 cps component of the EEG (a band-pass filter for quantification of this component—Ross & Davis, 1958—was included in this study) also show a drop in

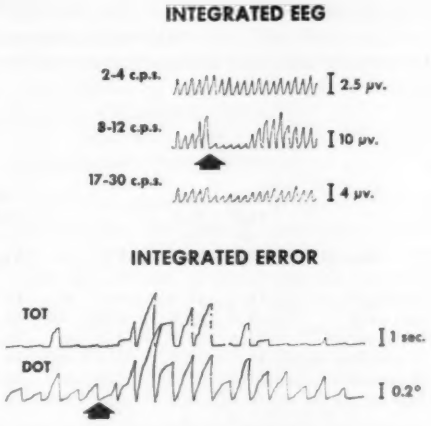


FIG. 4. Further data obtained from the same *S* as the one whose tracings are shown in Fig. 3. (The upper part of the figure shows the secondary EEG tracing consisting of the 4-sec. integrated outputs of three band-pass filters with the following frequency bands, from top to bottom: 2–4 cps, 8–12 cps, and 17–30 cps. The lower part of the record shows the sudden increase in error during the period when the *S* was dozing. Top line: 4-sec. integrations of TOT. Bottom line: 4-sec. integrations of DOT. The two arrows in this figure and the one in Fig. 3 indicate the same moment in time.)

amplitude (third line), while the 2–4 cps component appears to show no change. The integrator deflections for TOT (fourth line) and for DOT (fifth line) give evidence of a sudden increase in error that corresponds rather closely with the drop in amplitude of the EEG deflections. Because of the phase of the tracking cycle where the onset of sleep occurred, nothing should be made of the fact that the fall in EEG amplitude precedes the abrupt increase in error by several seconds. Heart rate (not included in the figure) showed a corresponding decrease during this brief period of sleep, followed by a rise.

*Heart Rate*

Mean heart rate for $S_1$ and $S_3$ was reliably higher (.001 level) in the second half of the vigil compared with the mean value for the first half of the vigil. The rises

TABLE 3

CHANGES IN MUSCLE TENSION DURING VIGIL

| Muscle | $S_1$ Mean Microvolts 1st Half of Vigil | $S_1$ Mean Microvolts 2nd Half of Vigil | $S_1$ Diff.[a] | $S_1$ $p$ | $S_2$ Mean Microvolts 1st Half of Vigil | $S_2$ Mean Microvolts 2nd Half of Vigil | $S_2$ Diff. | $S_2$ $p$ | $S_3$ Mean Microvolts 1st Half of Vigil | $S_3$ Mean Microvolts 2nd Half of Vigil | $S_3$ Diff. | $S_3$ $p$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Forehead | 18.5 | 17.9 | −0.6 | *ns* | 18.8 | 11.0 | −7.8 | <.001 | 9.3 | 10.8 | 1.5 | <.01 |
| Left neck | 8.9 | 14.9 | 6.0 | <.001 | 41.0 | 48.6 | 7.6 | *ns* | — | — | — | — |
| Right biceps | 42.6 | 29.9 | −12.7 | *ns* | 26.3 | 33.8 | 7.5 | <.01 | — | — | — | — |
| L. pronator teres | 11.0 | 5.9 | −5.1 | <.001 | 10.5 | 4.3 | −6.2 | <.001 | 12.4 | 8.7 | −3.7 | <.05 |
| R. pronator teres | 60.7 | 61.8 | 1.1 | *ns* | 76.9 | 41.9 | −35.0 | <.001 | 63.2 | 64.4 | 1.2 | *ns* |
| Left leg | 6.7 | 7.2 | 0.5 | *ns* | 6.6 | 5.9 | −0.7 | *ns* | 2.9 | 3.7 | 0.8 | *ns* |

[a] Plus differences signify rise, minus differences signify fall in muscle tension from first to second half of vigil.

were from 90.5 beats per minute to 103.8 beats per minute for $S_1$ and from 81.6 to 86.7 for $S_3$. $S_2$ showed an opposite trend, a drop from a mean heart rate of 84.0 beats per minute for the first half of the vigil to 79.6 beats per minute for the second half of the vigil. The difference met the .05 criterion of significance.

*EMG*

*Specificity of Tensional Increase during the Vigil.* Table 3 presents the muscle tension data fractionated for first and second halves of vigil. A difference prefixed by a minus sign represents a fall in muscle tension from the first to the second half of the vigil. Although the interpretation of the EMG findings is hampered by the absence of data from neck and biceps muscles of $S_3$, inspection of Table 3 reveals an interesting pattern in the results. All *S*s showed a significant increase of tension in only one muscle. The particular muscle concerned varied from *S* to *S*: $S_1$ showed increased tension in the neck muscles, $S_2$ in the right biceps, and $S_3$ in the forehead muscle.

The neck muscle data from $S_1$ are plotted in Fig. 5. It will be seen that the steepness of EMG gradient as well as the level of muscle tension increased progressively throughout the vigil. As would be expected, the dotted line curve for the single (high-incentive) trial at the end of the vigil has a less regular appearance than the other curves; but it is of interest to note that although this curve has a steeper slope than the other curves, the level of muscle tension appearing at the end of this trial was approximately the same as the final value of tension in the curve based on mean values from the last three sessions.

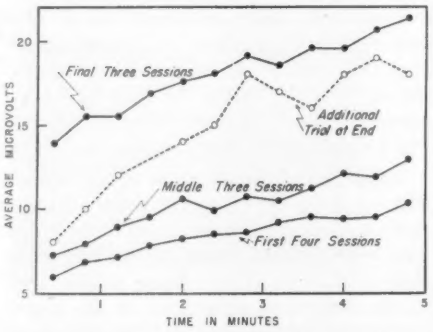


FIG. 5. Rise in neck muscle tension in $S_1$ during the vigil. (Data are plotted to permit visualization of mean EMG gradients for approximate thirds of the vigil and for final trial at the end of the vigil.)

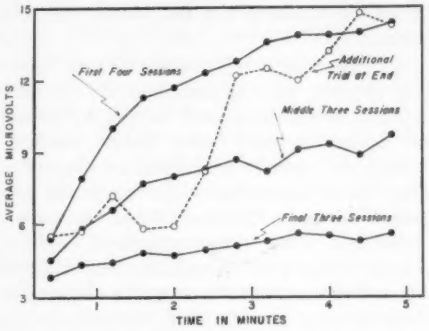


FIG. 6. Mean fall in left arm tension during the vigil. (Data are plotted in the same way as in Fig. 5, but data for all three *S*s are included.)

*Progressive Fall in Left Pronator Teres Muscle Tension during the Vigil.* Figure 6 presents curves for left pronator teres EMGs. These curves were plotted in the same manner as those shown in Fig. 5, but in this instance the mean muscle potential values plotted were taken from all three *S*s. As in Fig. 5 the curves show a progressive change in gradient steepness and in muscle tension level throughout the vigil, but the direction of change is exactly opposite to that shown in Fig. 5. There was continuous fall in level of tension and in gradient slopes for the left pronator teres muscle throughout the vigil. It should be noted that on the special incentive trial at the end of the vigil, mean tension and gradient steepness agreed very well with the corresponding mean values for the first four sessions. That the decrease of tension in this muscle was reliable in the case of each *S* may be seen by reference to Table 3.

### *Performance Data*

Table 4 presents the data on tracking performance for each *S*. Amount of error for the first half of the vigil is compared with the amount of error for the last half. It is clear from Table 4 that the three *S*s were rather different with respect to the effects of the vigil upon their performances. $S_1$ showed clear impairment; the increases in TOT and DOT were both highly significant. On the other hand, $S_2$ showed an improvement in his TOT score that was also highly reliable. This *S*'s DOT score showed no change, however. Finally, $S_3$'s change in TOT was not significant although his rise in mean error score in degrees met the .05 criterion of reliability. In summary, $S_1$ showed definite impairment, $S_3$ only slight impairment, while $S_2$ actually showed improvement in performance during the second half of the vigil compared with performance during the first half.

### *Subjective Reports Concerning Tracking Sessions*

*Hallucinatory Activity.* At the end of the last (tenth) tracking session, in his own

TABLE 4

CHANGES IN TRACKING PERFORMANCE DURING THE VIGIL

| | $S_1$ | | | | $S_2$ | | | | $S_3$ | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1st Half of Vigil | 2nd Half of Vigil | Diff.[a] | *p* | 1st Half of Vigil | 2nd Half of Vigil | Diff. | *p* | 1st Half of Vigil | 2nd Half of Vigil | Diff. | *p* |
| Mean TOT (sec./trial) | 64.8 | 120.8 | 56.0 | <.001 | 98.2 | 86.9 | −11.3 | <.001 | 109.4 | 105.2 | −4.2 | *ns* |
| Mean DOT (degrees) | 0.18 | 0.31 | 0.13 | <.001 | 0.16 | 0.16 | 0.00 | — | 0.25 | 0.27 | 0.02 | <.05 |

[a] Plus differences signify rise, minus differences signify fall in error scores (improvement) from first to second half of vigil.

handwriting $S_1$ wrote the following account of hallucinatory activity.

[I noticed] a funny effect of [the] tone in [the] last session. Often I would completely forget the task, and the tones would become part of something like a dream. Different discrete tones (from switching the knob around) became people! And there were patterns of them: rows and squares, which were interesting but annoying, since I wanted to keep attention only on the tracking.

On the next day following a good night's sleep in the laboratory, the $S$ added to his account as follows:

I would have the feeling of moving, making my way among the "people" at this time, with no auditory sensations at all. I think during these times I was still moving the knob because Dave didn't say anything to wake me up.

As a psychologist who realized that under unusual conditions (e.g., perceptual isolation) hallucinations could appear in nonpsychotic individuals, $S_1$ was able to view these experiences rather objectively, and hence did not seem much disturbed by them.

$S_2$ did not have any visual hallucinations but he did report a loss of ability to appreciate the true shape of the knob by tactual and kinesthetic means. During one trial he just "blinked" at it to get a glimpse of the knob. He said that it was disturbing not to be getting sufficient information from kinesthetic and cutaneous senses. This loss of cutaneous appreciation of the knob apparently occurred mainly during Session 5 when the rotational action of the knob had become somewhat stiff. It seemed to the $S$ that during this time he exerted considerably more pressure on the knob than at other times.

$S_3$ reported hallucinations which were inanimate, and were a materialization of a visual schema that he had been employing as an aid in tracking. He visualized a rectangle which represented the error-free zone in tracking. Inside this rectangle he visualized a black dot which represented the position of the brush that he could move by means of the knob that he held in his right hand. In his mind's eye he visualized the rectangle as moving from right to left as tracking commenced, and his self-instructions were to turn the knob in such a way as to keep the dot within the rectangle.

In the practice trials when he was learning to track, the mechanism of the tracking apparatus was explained to him by means of a diagram, and being "visual minded" (the $S$ customarily used visual aids in studying for examinations), he projected the visual image as if seen on a piece of paper. Although these images appeared to have clarity verging on the eidetic they differed in certain important respects from the hallucinatory images that occurred in the later sessions. Unlike the hallucinations, the visual images used as guides had definite form and they were completely under control.

The transition from the stage of imagery used as visual aid to hallucinations was a smooth one. Apparently the hallucinations first appeared briefly in Session 8 but did not develop fully until Session 9. The $S$ began to lose control of his imagery and the definite form of the images was replaced by undulating, wavy, irregular borders without sharp angles. The hallucinated figures were two-dimensional ones, projected some distance from the eyes as if on a movie screen. In his notes the $S$ wrote as follows:

The test [Session 9] began in much the usual way, but during trial no. 3 or no. 4 the visual image I have of the tone and which I use in tracking was replaced by indistinct hallucinations—halves of large objects—and the tracking test became a test of outlining these objects. I was rather surprised to note that my score on the first such test, trial no. 3 I think, was as good as it was, or as [the $E$] said it was. Deep respiration seemed to overcome or help me to dispel these mental imageries, and it (deep breathing) had to be maintained voluntarily and with considerable concentration in order to permit me to visualize the test pattern as I have been used to.

Further questioning revealed that for a session or two the $S$ was preoccupied with what he thought was a connection between the hallucinations and breathing. This even developed for a brief time into a delusion that the hallucinations were being induced by the $E$s. He thought that he noted some connection between the disappearance of the hallucinations and "disturbances" in the control room where the $E$s were operating

the equipment. By "disturbances" the *S* meant talking. The *S* had the feeling that his deep breathing was undesirable from the point of view of the *E*s (because of the artifacts produced in the physiological recordings), but he stated in his notes that he continued to breathe deeply despite this. Careful examination of the respiratory tracings revealed very little actual deep breathing during tracking trials, although increased amplitudes of the respiratory tracings were noted in the rest periods between trials in Session 9, and later in this session the pattern of respiration which had previously been very regular became irregular. It is likely, therefore, that the *S* exaggerated the amount of deep breathing that he engaged in.

Again from the *S*'s notes there were the following statements made immediately following Session 9 when the hallucinations were at their peak:

During the tracking there were gross tremors which later became smaller to fine, and which are only now disappearing; also there was excess salivation.

After the final session (10) the *S* wrote as follows:

I put maximum effort into concentrating on sounds alone, in an effort to reduce the number of hallucinations, and I very seldom allowed myself consciously to project a visual image of the sound. I saw only a few hallucinations which were similar to those described above; when they occurred they were transitory and my surprise or fear of seeing them was changed as a result of the [explanation of how hallucinations may be produced in normal individuals under unusual circumstances].

The *S* emphasized that he seemed to be able to ignore the hallucinations in this session by concentrating on the sound only.

It is interesting to note that roughly six weeks later when this *S* was interviewed again there appeared to be some confusion in his mind about the details of the visual schema that he had used during some nine hours of tracking during the vigil.

*Rating Scales.* Figure 7 presents the data from the rating scales for effort ("How much effort did you have to put in the task in order to do as well as you could?") and feeling of fatigue ("How do you feel now: very fresh, fresh, as usual, tired, very tired?"). The curves, which represent mean values for the three *S*s, show the expected changes in the direction of increased effort, and presence of a tired feeling in the latter part of the vigil. $S_3$ appeared to show a "second wind" phenomenon during the sixth session which is reflected in the group curve. Examination of the physiological and performance data did not confirm this finding. Except for a steeper EMG gradient for the left pronator teres muscle during this session, there were no conspicuous differences. The gradient in the right pronator teres was quite low, and there was nothing to indicate a break in trend in the data for heart rate, respiration, palmar conductance, and the error scores (DOT and TOT).

*Further Subjective Reports from Protocols.* $S_1$ found it more difficult to stay awake during the tracking sessions than at any other time. Early in the vigil he stated that he was bored and found it more difficult to overcome boredom than to stay awake. As the vigil continued to the final tracking ses-

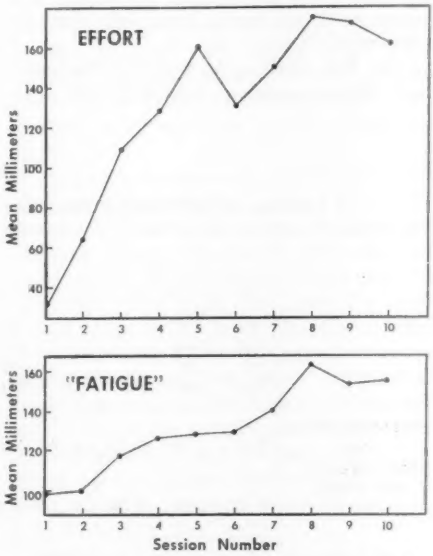


FIG. 7. Mean rise in ratings of effort and feeling of fatigue.

sion, however, his notes indicate that it was increasingly difficult to stay awake and that he was constantly fighting sleep. Verbatim notes from Thursday at 4:15 A.M.:

> Very hard to stay awake during tracking and almost impossible to keep eyes shut in between trials: It's either open them or sleep.

This *S* had the impression that by the eighth tracking session he was "getting lost" in the second half of the trials. Analysis of the error data, however, failed to support this subjective impression.

From his notes, it appeared that $S_2$ was more conscious of drowsiness when he was not occupied (as during lead placement and rest periods). When tracking commenced his feelings of drowsiness were reduced. $S_2$'s notes reflect a strong desire for high achievement in this task. He wrote that his performance had "deteriorated" during Session 5, but analysis of the error scores revealed that mean error score for this session was actually lower than the scores had been in the previous four sessions. Following Session 8 he wrote:

> It seemed that even though my thoughts were elsewhere, I was able to track automatically to a greater or lesser degree, being only minimally conscious of the tone.

In the last sessions $S_2$ reported "catnaps" and discouragement regarding his performance, but analysis of the error data revealed that he overestimated the effects of sleep loss on his performance.

Except for the reports on hallucinations, there was little else of interest in the notes from $S_3$.

### *Pain Thresholds*

In each case the mean pain threshold for the last day of the vigil was lower than the mean pain threshold for determinations made prior to the last day, and following sleep after the vigil. The mean thresholds in watts were 360 and 401 for $S_1$ (difference of 41 significant at the .02 level), 280 and 290 for $S_2$ (difference of 10 not significant), and 245 and 277 for $S_3$ (difference of 32 significant at the .001 level).

### *Ataxiameter Data*

The ataxiameter data in Table 5 show that body sway was significantly increased in the second half of the vigil in the case of $S_1$, significantly decreased in the case of $S_2$, and not significantly changed in the case of $S_3$. The decrease in sway for $S_2$ was significant for one measure (maximal shift) at the .02 level, and the decrease by the other measure (mean shift per 10 sec.) barely missed significance at the .05 level.

TABLE 5

CHANGES IN BODY SWAY AS MEASURED BY THE ATAXIAMETER DURING THE VIGIL

| | $S_1$ | | | | $S_2$ | | | | $S_3$ | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1st Half of Vigil | 2nd Half of Vigil | Diff.[a] | *p* | 1st Half of Vigil | 2nd Half of Vigil | Diff. | *p* | 1st Half of Vigil | 2nd Half of Vigil | Diff. | *p* |
| Mean shift/10 sec. (cm.) | 0.57 | 1.30 | 0.73 | <.02 | 0.31 | 0.23 | −0.08 | *ns*[*] | 0.22 | 0.26 | 0.04 | *ns* |
| Mean maximal shift/2 min. (cm.) | 4.93 | 8.91 | 3.98 | <.05 | 3.30 | 2.04 | −1.26 | <.02 | 2.48 | 2.26 | −0.22 | *ns* |

[a] Plus differences signify increase, minus differences signify decrease in amount of body sway from first to second half of vigil.

[*] $t = 2.06$, $t_{.05} = 2.07$.

## Discussion

### *EEG*

The decrease in amplitude of the alpha component of the EEG observed with progressive sleep loss in this study is consistent with the findings of previous investigators. The methods used in quantifying the EEG data have, of course, differed from investigator to investigator. Tyler et al. (1947) used manual methods of analysis and found a decrease in the 9–10 cps band. Bjerner (1949) also used manual methods but classified frequencies of approximately 10 cps as alpha activity in the case of three of his *S*s and 8.5 cps and 7.5 cps, respectively, in the case of the other two (pp. 58–59). Armington and Mitnick (1959), on the other hand, used an automatic digital-type method with time as the unit of measurement. In this case, the frequency band 8–12 cps was quantified and EEG activity was considered to be present only when the amplitude of the activity in this band exceeded 25 $\mu$v. (with 2 *S*s, 15 $\mu$v. was the criterion). Despite these obvious differences, however, there has been (together with the present results) substantial agreement in the finding that EEG activity in the "alpha" band has decreased with prolonged deprivation of sleep.

Such agreement does not, however, extend to the interpretation of these findings. Tyler et al. concluded that their results were associated with the effort of the *S*s to stay awake. Bjerner, on the other hand, concluded quite reasonably that his own findings of decreased alpha activity during sleep loss were associated with brief periods of sleep. Finally, Armington and Mitnick interpret their findings of low alpha during sleep deprivation as being due to drowsiness.

In previous articles (Malmo, 1958, 1959), some of the data reported in the present experiment were dealt with in relation to activation theory. To summarize the main points previously made, depriving an organism of sleep appears to be like depriving it of other basic needs. Under conditions of sufficient environmental stimulation this deprivation leads to increased activation. That is, judging from the overall picture of physiological change during our experimental vigils, it was concluded that the tracking task was sufficiently stimulating to interact positively with deprivation to produce a significant rise in activation during the 60-hour vigil.

It is clear that a difficult problem of interpretation has been raised by these different studies. Before returning to a consideration of the separate studies, it will be useful to recognize one very important general point. This is simply that low alpha EEG activity can, when it is observed, indicate either a high level of alertness (high activation) or a drift toward sleep (low activation). This to be sure is not a new finding—it has been reported in numerous studies. Only recently, however, have the implications of this finding for behavioral studies been made explicit (Malmo, 1959; Stennett, 1957a). We may conclude then that, if we consider only the level of alpha EEG activity at a given time—and nothing else—the question of whether an *S* shows high or low activation is indeterminate. In the light of this conclusion it is obvious that the apparent issue of interpretation which has been raised reduces to a matter of other evidence. It revolves around the question: Besides level of EEG alpha, what *other* evidence has been brought to bear on the matter? Let us examine the various studies in turn with reference to such additional evidence.

Although no measures other than the EEG were employed by Tyler et al. (1947) to assist with the interpretation of the alpha findings, these workers did show that accompanying the fall in 9–10 cps activity there was a rise in the fast-frequency component of the EEG (i.e., a higher percentage of waves in the 11–17 cps band, and in the band from 18 cps and higher). This certainly is more consistent with the interpretation of increasing activation rather than the reverse inasmuch as a decrease *not* an *increase* in the higher frequencies is to be expected with a change in the direction of sleep (Lindsley, 1957, p. 68).

Bjerner (1949), it will be recalled, did not deal with long-term changes in alpha but with short-term, transient changes during

"delayed actions." His finding of decreased alpha was associated with evidence from behavioral observations as well as evidence from another physiological measure (heart rate). The EEG changes he observed were accompanied by overt signs of sleep (marked delay in response to a signal, snoring) along with a reliable fall in heart rate. From this additional evidence it seems clear that Bjerner's *S*s were at these times drifting down the activation dimension.

It is unfortunate that Armington and Mitnick (1959) presented no physiological evidence besides the 8–12 cps EEG data; but it seems likely that they are correct in interpreting their findings of low alpha during sleep deprivation as being due to drowsiness. In other words, it appears probable that they were unsuccessful in their attempt to investigate the effects of sleep deprivation under conditions less favorable for dozing than in previous studies: having their *S*s stand was certainly no guarantee that the *S*s would not drift toward sleep.

To reiterate, in our experiment the EEG and other physiological findings are consistent with the interpretation that our *S*s were alert and in a high state of activation. We are not aware of any instance where rise in palmar conductance or respiration rate has been reported to coincide with a drift down the activation dimension toward sleep.

Because so little has been reported about the 2–4 cps component of the EEG in waking *S*s it is difficult to interpret the finding that tracings from $S_1$ and $S_3$ showed a significant fall in amplitude. However, two rather general comments may be made. Firstly, it is interesting to note that once again it was $S_2$ who was the exception. Secondly, $S_1$ whose tracking performance deteriorated more than the others showed the greatest fall in 2–4 cps EEG. If $S_1$'s drop in performance were interpreted as due to drift toward a sleepy state, it is likely that, if anything, a rise in amplitude of frequencies in this low range would have been expected instead of the fall that was observed (Malmo, 1959, p. 380). The same general argument holds for $S_3$ who showed a slight drop in performance. $S_2$, it will be recalled, actually improved in tracking in the second half of the vigil so that the slight rise in 2–4 cps EEG amplitude can hardly be accounted for in terms of increasing drowsiness in this *S*.

*Other Physiological Measures*

*Heart Rate.* $S_1$ and $S_3$ showed significant rise in heart rate during the vigil. This finding was consistent with the rise in palmar conductance and in respiration rate. The results from $S_2$, on the other hand, were the exact opposite; in his case there was falling heart rate which on the basis of a comparison between first and last halves of the vigil was statistically significant.

This reversal in heart rate is difficult to interpret considering that the palmar conductance, respiration rate, and 8–12 cps EEG data were very similar to those of the other two *S*s (see Table 2). However, we may note that, in referring to the experiments of Hickam and his co-workers, Gellhorn (1953, p. 338) indicated that under activating conditions which produce heart rate acceleration in the majority of *S*s, some *S*s show diminished heart rate and lowered blood pressure. As a matter of fact, $S_2$ did show a fall in blood pressure from the first half to the second half of the vigil, and although the difference was not large it was significant (at the .01 level). $S_1$, on the other hand, showed a rise in blood pressure from the first half to the second half of the vigil, though this rise was not statistically significant. The attempt to record blood pressure from $S_3$ was abandoned because finger movement artifacts made it impossible to use this instrument (which depended on a finger pickup).

However, it should be made clear that there is evidence against the notion that $S_2$ simply reacts with a drop in heart rate under all conditions in which the heart rates of others increase. Data taken several months before his participation in the sleep deprivation experiments clearly showed typical heart rate acceleration in this *S* under certain activating conditions. Comparison of heart rate for 5-min. trials during the last half of the session with that for the 5-min. trials during the first half of the

session showed a significant rise in the second half.

Finally, it may be noted that even in the second half of the vigil despite the downward shift in the heart rate baseline, heart rate rose regularly at the end of each trial, during the time that the *S* was waiting for and receiving information concerning his performance on that trial.

*EMG.* As previously stated each *S* showed a significantly *increased* tension in one particular muscle (left neck, right biceps, and forehead, for $S_1$, $S_2$, and $S_3$, respectively), and all *S*s showed a progressive *decrease* in level and in steepness of gradient of EMGs from the left pronator teres muscle during the vigil (see Fig. 6).

The findings of increased muscle tension localized in one muscle group has been discussed previously in relation to the specificity of localized muscle tension in psychiatric patients with complaint of discomfort from these tense muscles (Malmo, 1959). The previous statement was as follows:

> Where high level activation is long continued as in a vigil or in certain psychoneurotic patients, it appears that skeletal tension may become localized to a single muscle group. The discomfort associated with this tension in some patients can become extremely severe. It should be noted that in one-session experiments, where rise in activation was for relatively short intervals of time, tensional rise occurred in more than one muscle group.
>
> Methodologically, these results are important because they reveal a difference between EMGs and some other physiological measures with respect to gauging activation. Unlike heart rate or respiration rate that invariably yields one measure no matter how it is recorded, there are as many measures of muscle tension as there are muscles that can be recorded from. It appears that when sufficient care is taken, EMGs may be very valuable in helping to gauge activation, but that considerable caution is required in the interpretation of results, and especially in the interpretation of negative results (p. 383).

At the very end of the vigil each *S* was given an extra 5-min. trial, after being informed that this was his *very* last tracking trial. Under these conditions it was interesting to note that the tension in the left pronator teres muscle that had been gradually decreasing all through the vigil, returned to the level it had reached during the early trials and also showed approximately the same steepness of gradient. This finding suggests that there might have been something like a conservation of energy mechanism operating to reduce the tension in the left arm during the vigil, and that when the vigil was over at last, and such conservation of energy was no longer required, the tension in the left arm returned to its previously higher level. A puzzling question is why, if such a conservation mechanism existed, it did not operate on all of the muscles. Of course with a larger group of *S*s and with more channels of EMG it is quite possible that variations would be found, with some individuals showing rise in more than one muscle, and some individuals perhaps not showing a rise in any muscle. However, considering the small number of *S*s in this investigation, the consistency of the findings was remarkable.

*Pain Thresholds*

The three *S*s were consistent in the direction of change (a drop) in pain threshold with sleep deprivation, although $S_2$'s change was not statistically significant. These findings are on the whole consistent with those of Cooperman et al. (1934) whose report was summarized in the introductory section of this paper. Although the negative results of Schumacher et al. (1940) are occasionally cited as evidence against a change in pain threshold with sleep deprivation, this does not appear to be a fair conclusion because the threshold determinations were carried out after only 24 hours of sleep deprivation. Actually the main purpose of the experiments by Schumacher and his coworkers was to test the influence of a rather moderate amount of sleep deprivation on the pain threshold.

As far as we know, pain is the only modality that has been found more sensitive under conditions of sleep deprivation. In the introductory section we briefly summarized the findings concerning other sense modalities. From this evidence it appears that pain is unique with regard to the effect sleep deprivation has upon it. This uniqueness is of interest in relation to Hebb's (1949) theory of pain. Hebb emphasizes

the importance of central mechanisms in the mediation of pain intensity, and lays heavy stress on high-level discharges in the central nervous system. According to the view that sleep deprivation increases level of activation, it would follow that if Hebb is correct, and if pain does depend more on quantity of central discharge than the other modalities, the pain threshold should be lowered by sleep deprivation. The reasoning is as follows. Assume that the threshold for pain requires a certain level of firing in a particular part of the brain, and that this part of the brain receives impulses from the activating system. Sleep deprivation increases the contribution from the activating system with the result that the critical (threshold) value is reached sooner.

Our findings of decreased pain threshold were obtained without the concomitant measurement of physiological functions. It is not possible to state with the same degree of confidence as for tracking, that activation level increased from first to last threshold determination, although this seems a reasonable assumption. Careful study of pain threshold changes in relation to shifts in activation level (as gauged by physiological indicants) appears to be an area in need of research. Vernon's (1959) findings of lowered pain threshold in sensory deprivation experiments, for example, could be followed up in this manner; and it may also be noted that this finding seems to fit Hebb's hypothesis that pain is potentiated in some cases by *de*creased *peripheral* sensory activity.

*Performance*

In tracking, $S_1$ showed marked impairment, $S_2$ showed significant improvement, while $S_3$ showed very slight net impairment It was argued earlier in this paper that the incentive conditions were effective in keeping alertness at a high level throughout the vigil, and the general picture of increased activation shown by the physiological data was offered as evidence that the incentive conditions were indeed effective. Now, the fact that $S_2$ actually improved in performance during the vigil and $S_3$ showed only very slight impairment on one of the two error measures is further evidence for the effectiveness of these incentives.

Possibly the most difficult interpretation is that of $S_1$'s performance. Does a large drop in tracking performance fit in with the conclusions of increased activation? In retaining the conclusion of increased activation, and not considering other complications, we are forced to interpret the impaired performance as being due to overactivation. There are, of course, a number of other factors that must be considered. It could be argued, for example, that $S_1$'s hallucinatory experiences might have impaired performance during the later sessions when they occurred, and the absence of such experiences in the case of $S_2$ might have had something to do with his success.

It is unlikely that $S_2$'s improvement was due to practice during the vigil since, as previously mentioned, he had had a considerable amount of practice with the tracking apparatus during an exploratory experiment that was conducted several months before the sleep deprivation experiment, and in addition had been brought up to a practice level on tracking just prior to the sleep deprivation experiment.

*Ataxiameter Performance.* If standing still (resisting sway) is viewed as a task or an act of skill then it may be said that sleep deprivation produced very much the same changes in this task as it produced in tracking. Thus, just as in the case of tracking performance $S_1$ showed significant impairment, $S_2$ showed significant improvement, while $S_3$ showed only slight insignificant changes during the vigil.

*Subjective Reports concerning Tracking.* The curves plotted from the rating scale data showed that the *S*s' estimates of fatigue, and of effort expended in tracking, were in fair agreement with the physiological data but not with the performance data. For instance, $S_1$ and $S_2$ rated themselves on effort and fatigue in very much the same way despite the fact that their tracking performances took opposite directions during the vigil.

Furthermore, there appeared to be rather poor agreement between the comments of the *S*s with respect to how they felt and how they actually performed on tracking. For instance, although $S_2$ reported "catnaps" during Session 9, and feared that sleep loss might spoil his good tracking record, his performance during this session turned out to be even better than it had been earlier in the vigil.

It may be significant to recall that $S_1$ spoke of being confused in the later trials when his performance was showing gross impairment, whereas $S_2$ in contrast with this, stated that he could even let his thoughts wander a little away from the task without letting his performance suffer. It must be pointed out, however, that $S_1$'s statements about confusion appeared to be connected with efforts to fight off sleep, and he too mentioned dozing a few times. On the face of it, this does not fit with the notion of $S_1$'s impairment being due to overactivation; however, it should be recognized that the overactivation interpretation in his case must be regarded as tentative. Actually, there is rather good evidence from animal studies that overactivation can occur with sleep loss. In an unpublished study by Ehrlich in our laboratory, certain rats appeared greatly overactivated, occasionally displaying what appeared to be confused excitement in which, for example, the animal attacked the bar, biting it, instead of pressing for food reward as it previously had done. Licklider and Bunch (1946) reported that sleep-deprived rats became so aggressive they had to be kept separated, because if allowed to come together they fought viciously, killing one another.

*Hallucinatory Experiences*

The hallucinatory experiences in two of the three *S*s, and the delusional experiences in one, were in their clarity, and other ways, different from related experiences reported in other investigations of sleep deprivation.

Although reviews of sleep-deprivation experiments usually mention hallucinatory experiences as a common finding there have been in fact very few well described experiences of this kind. With the exception of the large-scale study by Tyler (1947) and the recent studies by Bliss, Clark, and West (1959) and by Morris, Williams, and Lubin (in press), references to such experiences, when they were made at all, were rather vague.

It is probably significant that the hallucinatory and delusional experiences observed by us were very closely connected with the tracking task. The fact that during tracking sessions the *S*s sat with eyes closed for an hour or more may be related to the visual character of the hallucinations. As far as we know, the conditions of other sleep-deprivation experiments did not include such long periods of working without vision. Other workers who recorded EEGs, such as Bjerner (1949) and Armington and Mitnick (1959), also required the *S*s to work on tasks with eyes closed, but the periods of work were considerably shorter than 1 hour and, as far as we know, task-related hallucinations did not occur in either of these studies.

The presumed hallucinogenic effect of excluded vision in our experiments may be related to the hallucinatory effects produced in experiments of perceptual isolation (Bexton, Heron, & Scott, 1954; Hebb, 1958; Heron, 1957; Heron, Doane, & Scott, 1956). It is of interest that the hallucinations in our experiments were confined to the tracking sessions and did not occur between these sessions when the *S* was presented with a variety of stimulations, visual and otherwise. It seems a reasonable speculation, therefore, that the exclusion of vision, plus the constant attention to the tones in the earphones, may have interacted with sleep deprivation to produce the hallucinations.

That sleep deprivation may interact with other conditions to produce hallucinations is also suggested by the work of Bliss et al. (1959) on LSD–25. From their experiments it was concluded that sleep deprivation may react with LSD–25 to produce hallucinations with doses that had previously been found nonhallucinogenic in the same *S*s when not sleep deprived.

## SUMMARY AND CONCLUSIONS

Three separate 60-hour vigils were held with each of three healthy young male *S*s. During each vigil, 10 1-hour recording sessions were held several hours apart. In each session the following measurements were recorded continuously while the *S* was performing on a tracking task with 5-min. trials and 1-min. rests: EEG, EMGs from various muscles, respiration, palmar conductance, and heart rate.

Main findings were that in all three *S*s, palmar conductance and respiration showed a statistically reliable increase, and 8–12 cps EEG a reliable decrease in amplitude. Heart rate was reliably increased in two of the three *S*s, (though reliably decreased in the third *S*). In addition, each *S* showed reliably increased muscle tension in only one muscle group (a different one in each *S*).

On the whole the data strongly supported the conclusion that sleep deprivation had the effect of increasing level of activation. In this respect the present investigation appeared to be different from certain other investigations of sleep deprivation in which conditions were not sufficiently alerting to prevent the *S*s from drifting toward sleep. With the overall indication of increasing activation from the peripheral measures, the fall in 8–12 cps EEG was interpreted as also indicative of increasing activation, and not as drift toward sleep.

Under conditions of this investigation, increased muscle tension appeared to be localized in one muscle group rather than generalized over the entire body. In fact, tension in some parts of the body actually fell progressively during the vigil.

It appeared that the special conditions of the tracking sessions were particularly conducive to the appearance of hallucinatory experiences.

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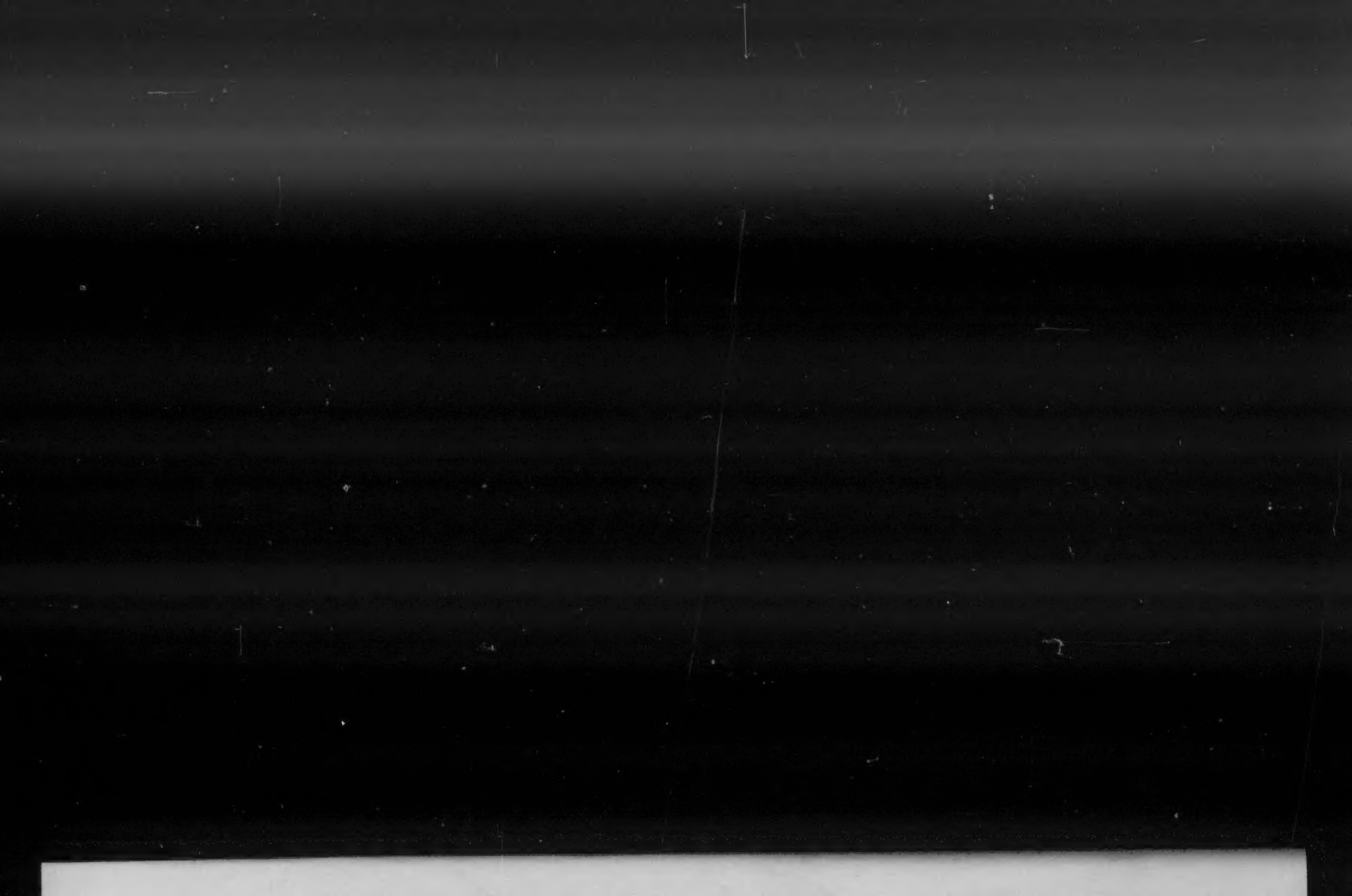
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